REMARKS

Reconsideration and withdrawal of the rejections of the claims, in view of the amendments and remarks herein, is respectfully requested. Claims 1-4, 6-15, 23, and 26 are amended. Claims 1-28 are now pending in this application. The amendments are intended to advance the application and are not intended to concede to the correctness of the Examiner's position or to prejudice the prosecution of the claims present prior to amendment, which claims are present in a continuation of the above-identified application.

Claims 1-4, 6-18 and 20-28 were rejected under 35 U.S.C. § 112, second paragraph, for indefiniteness. The amendment to claims 1 and 15 to delete "rodent" obviates this rejection.

Claims 1-4, 6-18 and 20-28 were rejected under 35 U.S.C. § 112, first paragraph, as lacking adequate enablement. In particular, the Examiner asserts that not all rodents, all Abetas from different species and all neurological diseases are enabled. This rejection, as it may be maintained with respect to the pending claims, is respectfully traversed.

As amended, the claims do not recite "rodent" or "neurological disease".

With regard to Abeta, please consider that the amyloid precursor protein (APP), which is the unprocessed precursor of Abeta, is highly conserved (see, e.g., the abstract for Yamada et al., BBRC, 149:665 (1987); a copy is enclosed herewith). Therefore, Applicant's disclosure that perfusion of a rat with Abeta, in conjunction with other agents, results in a rat with impaired performance in memory and learning and having hyperphosphorylated tau, amyloid plaques or neurofibrillary tangles, enables the use of Abeta generally to prepare such rats.

Accordingly, withdrawal of the § 112 rejections is respectfully requested.

CONCLUSION

Applicants respectfully submit that the claims are in condition for allowance and notification to that effect is earnestly requested. The Examiner is invited to telephone Applicants' attorney (612) 373-6959 to facilitate prosecution of this application.

If necessary, please charge any additional fees or credit overpayment to Deposit Account No. 19-0743.

Respectfully submitted,

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Date September 3, 2008

By //Janet E. Embretson//
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CERTIFICATE UNDER 37 CFR 1.8: The undersigned hereby certifies that this correspondence is being filed using the USPTO's electronic filing system EFS-Web, and is addressed to: Mail Stop Amendment Commissioner of Patents, P.O. Box 1450, Alexandria, VA 22313-1450 on this 3rd day of September 2008.

Amy Moriarty
Name

Signature

Complementary DNA for the mouse homolog of the human amyloid beta protein precursor.

Yamada T, Sasaki H, Furuya H, Miyata T, Goto I, Sakaki Y.

Research Laboratory for Genetic Information, Faculty of Medicine, Kyushu University, Fukuoka, Japan.

The human amyloid beta protein is a major component of brain amyloid found in patients with Alzheimer's disease. As an initial step to understand the biological function of its precursor protein, we have isolated cDNA for the mouse homolog of the human beta protein precursor. Comparison of the predicted amino acid sequence with that of human revealed a quite high degree of homology (96.8%), and the calculated evolutionary rate of the mRNA at amino acid substitution site was relatively low (0.1 x 10(-9)/site/year). The mRNA was abundant in brain and kidney, and also detected in other tissues at low level. These results indicated that this protein is highly conserved through mammalian evolution and may be involved in a basic biological process(es).

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